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Abstract

Parkinson's disease involves intracellular deposits of alpha-synuclein in the form of Lewy bodies and Lewy neurites. The alpha-Synuclein-containing aggregates represent a feature of a variety of neurodegenerative disorders, including Parkinson's disease. A lack of evidence supporting a role of heritability in the development of idiopathic Parkinson's disease (PD) has implicated environmental factors. Mutations in parkin are currently recognized as the most common cause of familial Parkinsonism. Emerging evidence also suggests a role for environmental factors. Extensive epidemiological data in humans and studies in animal models of Parkinson's disease (PD) suggest that sporadic Parkinson's disease is a multifactorial disorder. Environmental paraquat and neonatal iron exposure have both separately been suggested as potential risk factors for sporadic Parkinson's disease. Both genetic and environmental factors are thought to be involved in the aetiology of Parkinson's disease (PD). Oxidative stress and mitochondrial dysfunction have been frequently implicated in the neurodegenerative process that underlies Parkinson's disease. As Parkinson's disease appears to be a multifactorial disorder, the use of animal models to investigate combined effects of genetic and environmental factors is important. Parkinson's disease (PD) is one of the common neurodegenerative diseases that result in the progressive damage of dopaminergic neurons. Parkinson disease (PD) is a neurodegenerative disease characterized by death of dopaminergic neurons in the substantia nigra. Epidemiological studies indicate a role of genetic and environmental factors in Parkinson's disease involving alterations of mitochondrial function. Mitochondrial dysfunction has been frequently implicated in the neurodegenerative process that underlies Parkinson's disease. Parkinson's disease (PD) is characterized by loss of A9 dopaminergic (DA) neurons in the substantia nigra pars compacta. Parkinson's disease is associated with mitochondrial decline in dopaminergic neurons of the substantia nigra. One of the environmental risk factors for Parkinson's disease is exposure to pesticides. Paraquat (PQ) and maneb (MB) are able to induce neurotoxic effects by promoting alpha-synuclein (alpha-syn) aggregation. Paraquat (PQ) and maneb (MB) are able to induce neurotoxic effects by promoting alpha-synuclein (alpha-syn) aggregation. Parkinson disease is a debilitating and incurable neurodegenerative disorder affecting approximately 1-2% of people over 60 years of age. Epidemiological studies have suggested a correlation of pesticides and Parkinson's disease (PD) while genetic and biochemical studies have suggested a role for oxidative stress (OS). Oxidative stress (OS) stimulates autophagy in different cellular systems, but it remains controversial if this rule can be generalized. Cytoplasmic inclusions known as Lewy bodies, a hallmark of Parkinson's disease (PD) pathology, may protect against cytoplasmic stress. Currently, most neurotoxicological investigations are still conducted using various animal models (e.g. chickens, rodents). We examined effects of three structurally related pyridinium compounds, 1-methyl-4-phenylpyridinium (MPP+), paraquat (PQ) and maneb (MB) on PC12 cells. Apoptosis plays an important role in neurodegeneration, although the mechanisms and mediators in the brain are largely unknown. Paraquat was taken up by PC12 cells in a carrier-mediated, saturable manner. When PC12 cells were permeabilized with digitonin, paraquat uptake was increased. Although paraquat has been shown to cause oxidative damage to neuronal cells, little is known about its effect on glial cells. Drugs and certain environmental toxins may be responsible for the pathogenesis of Parkinson's disease. We have used pargline to study the effect of the induction of i-NOS in primary glial cultures was studied with respect to the protein levels of reactive oxygen species. Paraquat (PQ) is a well described pneumotoxigenic agent that produces toxicity by redox cycling with cellular diaphorases, thereby generating reactive oxygen species. The excitatory amino acid glutamate serves important neurologic functions, but overactivation of its N-methyl-D-aspartate (NMDA) receptor has been implicated in neurodegeneration. Various structurally unrelated chemicals [2,5 hexandione, acrylamide, organophosphates like mipafox, beta,beta iminodipropionitrile] have been shown to induce Parkinson's disease. Recent etiological study in twins (Tanner et al. 1999) strongly suggests that environmental factors play an important role in the pathogenesis of Parkinson's disease. Recent studies have demonstrated that inhibition of the proteasome, an enzyme responsible for the majority of intracellular protein degradation, leads to neurodegeneration. Treatment of neuroblastoma cells with the copper chelator triethylene tetramine tetrahydrochloride induced intracellular oxidative stress.

HumPrim	AniPrim	AniSec	InvAll	Does this article contain ADME data?
etiology of the disease is unknown, how			InvAll	
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forms of the d	AniPrim	AniSec	InvAll	
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SNpc). An association has been reporte			InvAll	
genes linked with the onset of Parkins			InvAll	
es and altering tyrosine hydroxylase (TH			InvAll	NOADME
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ells. Thus the effect of paraquat on glia			InvAll	NOADME
araquat as a model toxin for this study			InvAll	NOADME
gen species (ROS) scavenging enzymes			InvAll	NOADME
by elevating intracellular levels of supe			InvAll	NOADME
te (NMDA) receptor is toxic to neurons			InvAll	NOADME
propionitrile (IDPN), 3-nitropropionic			InvAll	NOADME
in typical, non-familial Parkinson's dise			InvAll	
ular proteolysis, may contribute to the			InvAll	
ir decrease of copper content parallele			InvAll	NOADME

Cell type (cell line, primary cells, recombinant protein)

Recombinant alpha-synuclein

Recombinant alpha-synuclein

PC12 cell line or primary ventral mesencephalic cultures

SH-SY5Y cell line

N27 cell line or E15 primary mesencephalic cultures

E14-15 primary mesencephalic neuron-glia and microglial cultures

SH-SY5Y cell line

N27 cell line or E15 primary mesencephalic cultures

PC12 cell line

BE2-M17 cell line or primary cortical neurons or primary astrocyte cultures

SH-SY5Y and SK-MEL-2 cell lines

Primary fibroblasts from PD patients and controls

Human induced pluripotent stem cells differentiated into dopamine neurons

E14-15 primary mesencephalic neuron cultures or C. elegans (HeLa cells also used but not considered a relevant cell type)

SH-SY5Y cell line

SH-SY5Y cell line

SK-N-MC cell line

astrocytoma U373 cells

SK-N-MCu cell line

Primary neuronal cell cultures

PC12 cell line

N9 microglial cells

PC12 cell line

C6 glioma cells

PC12 cell line

Glial cell cultures from the neocortex of newborn

CPA-47 endothelial cell line

Cortical neuron cell culture

nigral dopaminergic cell line, SN4741

SH-SY5Y cell line

SH-SY5Y cell line

Species	Exposure duration	Exposure details
Unclear	Up to 500 hours	
Unclear	Up to 40 hours	
Rat	6 to 48 hours	
Human	24 hours	
Rat or Mice	18 or 24 hours	
Mice	5 days or 10-30 minutes	
Human	48 hours	
Rat or Mice	24 hours	
Rat	12 hours	
Human or Mice	24 hours	
Human	72 hours	
Human	48 hours	
Human		
Mice or C. elegans	24 or 72 hours	
Human	24 or 48 hours	
human	24 hours	
human	24 hours to 1 week	
human	24 hours	
human	48 hours	
rat	3 and 7 days	
rat	6 hours	
mouse	4 hours	
rat	up to 5.5 hours	
rat	48 hours	
rat	48	
rat	8 hours	
cow	5 hours	
rat	7 days	
human	6, 12, 24, 48 hours	
human	12 and 24 hours	
human	24 hours	

Dose (uM)	alpha synuclein, Tau phosphorylation, tubulin	DA (TH+) neurons	Dopamine (DA and metabolite levels, DAT and receptor expression, TH immunoreactivity)	Cell viability (LDH levels, apoptosis, total cell number)
100	1			
10, 100, 500, or 1000	1			
10, 50, 75, or 100				1
1000 or 5000				
30, 100, 200, 300, or 400				1
0.5, 1, or 2		1		
300	1			1
30 or 350		1		1
50, 100, 200, or 500				1
2000 or 4000				1
150, 250, or 450	1			1
300	1			1
12.5 or 200		1		
50, 100, 250, 500, or 1000	1		1	1
100 - 500	1			1
0.01 to 10				1
100				
10, 50, 100				
1, 5, 10, 50, 100 ug/ml				1
0, 50, 100, 500				1
500				
250 or 1000				1
250, 500, 1000				1
500				1
50, 100, 500, 1000				1
0, 25, 75, 150, 300, 600				1
0.1, 1, 5, 10, 50 ug/ml	1			1
800		1		1
20, 200				1
500 or 1000				1

not relevant

[illegible]

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M. A. Moran Ortiz-Ortiz. Curcumin enhances paraquat-induced apoptosis of N27 mesencephalic cells via the generation

SH-SY5Y cells transfected with the enzymatically inactive Cu,Zn superoxide dismutase mutant H46R were more resistant to oxidative stress. Using the inactivation of mitochondrial and cytosolic aconitases as markers of compartment-specific superoxide ($O_2^{\cdot-}$) production, we investigated the effect of oxidative stress on neuronal death. Oxidative stress caused by an increase in free radicals plays an important role in neuronal death. We investigated the effect of oxidative stress on neuronal death.

PURPOSE: In a recent study, it was demonstrated that docosahexaenoic acid (DHA) promotes the survival of retinal photoreceptors. Oxidative stress has been discussed as crucial mechanism of neuronal cell death in the adult brain. However, it was not clear whether oxidative stress is supposed to play an important role in demyelinating diseases. Oligodendrocytes are the myelin-forming cells in the central nervous system. Paraquat (PQ; 1,1'-dimethyl-4,4'-bipyridinium dichloride) is widely used as a universal herbicide. Although systemic treatment with paraquat is highly toxic, we examined the toxicity of paraquat, a possible environmental risk factor for neurodegenerative disorders like Parkinson's disease.

We previously identified a novel endogenous substance, serofendic acid, from a lipophilic extract of fetal calf serum. Serofendic acid may play a role in oxidative stress. Oxidative stress is currently considered a mediator of cell death in several neurodegenerative diseases. Notably, it may play a role in the pathogenesis of Parkinson's disease. Oxidative stress has been implicated in neuronal death caused by cerebral ischemia or some neurologic disorders. Chemical-induced neuronal cell death induced by oxidative stress is correlated with numerous neurodegenerative diseases, including Alzheimer's disease and Parkinson's disease. Oxidative stress and apoptosis play pivotal roles in the pathogenesis of neurodegenerative diseases. We investigated the role of reactive oxygen species (ROS) in reperfusion injury after transient focal cerebral ischemia. The antioxidant capacity of the brain is an important factor in the pathogenesis of neurodegenerative diseases. Mechanisms involved in paraquat neurotoxicity that selectively target nigrostriatal dopaminergic neurons remain relatively unknown. The adaptive responses to H_2O_2 and the resulting protective effect against oxidative stress have been investigated using immortalized rat neuroblastoma cells. The exposure of immortalized rat neuroblastoma cells to MPP(+) and paraquat results in cell death. Heat shock pre-treatment with erythropoietin (Epo) expression, which regulates erythropoiesis, has been shown in rat and mouse brain after hypoxia. A number of studies have shown that reactive oxygen species (ROS) contribute to the development of various human diseases. Cu,Zn-superoxide dismutase (SOD) is a major antioxidant enzyme in the brain. Paraquat is a herbicide with a potential risk to induce parkinsonism due to its demonstrated neurotoxicity and its strong oxidative properties. Previous work demonstrated that a brief, sublethal excitotoxic insult strikingly increased the sensitivity of cortical neurons to oxidative stress. Mitochondrial damage is linked to many neurodegenerative conditions, such as Parkinson's disease, Alzheimer's disease, and Huntington's disease. Although reactive oxygen species (ROS) at physiological concentrations are required for normal cell function, excessive production of ROS is a major factor in the pathogenesis of neurodegenerative diseases. Identification of common mechanistic principles that shed light on the action of the many chemically diverse toxicants to the brain is an important goal. Parkinson's disease (PD) is characterized by selective loss of dopaminergic neurons in the substantia nigra of the brain. Autophagy is a degradative mechanism involved in the recycling and turnover of cytoplasmic constituents from eukaryotic cells. Paraquat ($PQ(2+)$) is a prototypic toxin known to exert injurious effects through oxidative stress and bears a structural similarity to the neurotoxin 1-methyl-4-phenylpyridinium ion. Previous studies have suggested that Ginkgo biloba extract (EGb761) has a protective potentiality against apoptosis of neurons. The JAK/STAT pathway is activated in response to cytokines and growth factors. In addition, oxidative stress can activate the JAK/STAT pathway. Excess production of reactive oxygen species (ROS) is an important mechanism underlying the pathogenesis of a number of neurodegenerative diseases. Transmissible spongiform encephalopathies are a group of neurodegenerative disorders caused by a posttranslational, conformational change in a protein. Green tea, owing to its beneficial effect on health, is becoming more and more popular worldwide. (-)-Epigallocatechin-3-gallate (EGCG) is a polyphenolic compound in green tea. Paraquat (PQ, 1,1'-dimethyl-4,4'-bipyridinium), a widely-used herbicide, has been suggested as a potential etiologic factor in the development of Parkinson's disease (PD). The herbicide paraquat is a suspected etiologic factor in the development of Parkinson's disease (PD). Paraquat was therefore suggested as a potential etiologic factor in the development of Parkinson's disease (PD). Paraquat (1,1'-dimethyl-4,4'-bipyridinium) is structurally similar to the neurotoxin 1-methyl-4-phenylpyridinium ion. In order to establish causal or protective treatments for Parkinson's disease (PD), it is necessary to identify the cascade of events leading to neuronal death. Hystidyl-proline [cyclo(His-Pro)] is an endogenous cyclic dipeptide produced by the cleavage of thyrotropin releasing hormone. XRCC1 is a critical scaffold protein that orchestrates efficient single-strand break repair (SSBR). Recent data has found an association between oxidative stress and Parkinson's disease (PD). Parkinson's disease (PD) features oxidative stress and accumulation of misfolded (unfolded, alternatively folded, or mutated) proteins. Paraquat (PQ) (1,1'-dimethyl-4,4'-bipyridinium dichloride), a widely used herbicide, has been suggested as a potential etiologic factor in the development of Parkinson's disease (PD). Oxidative damage is involved in triggering neuronal death in several retinal neurodegenerative diseases. The recent findings suggest that oxidative stress may play a role in the pathogenesis of Parkinson's disease (PD). The major advantage of primary neuronal cultures for developmental neurotoxicity (DNT) testing is their ability to replicate the pathogenesis of Parkinson's disease (PD). **BACKGROUND:** Mitochondrial dysfunction has been implicated in the pathogenesis of Parkinson's disease (PD). Impaired mitochondrial function is a common feature of Parkinson's disease (PD). The role of autophagy as a survival strategy of cells constitutes an emerging topic in the study of the pathogenesis of several neurodegenerative diseases. Mutations of the PINK1 gene are a cause of autosomal recessive Parkinson's disease (PD). PINK1 encodes a mitochondrial serine/threonine kinase. The precise mechanism underlying the role of nitric oxide (NO) or nitric oxide synthases (NOSs) in paraquat-mediated toxicity remains unclear. Curcumin, the active compound of the rhizome of *Curcuma longa* has anti-inflammatory, antioxidant and antiproliferative effects.

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Oxidative stress leads to the disruption of calcium homeostasis in brain neurons; however, the direct effects of oxidants c

Paraquat (1,1'-dimethyl-4,4'-bipyridinium), a widely used non-selective herbicide, is a redox cycling agent with adverse ef

Epidemiologic and laboratory studies suggest that paraquat can be an environmental etiologic factor in Parkinson's disea

BACKGROUND: Mitochondrial oxidative stress is a contributing factor in the etiology of numerous neuronal disorders. Ho

Exposure to environmental pesticides can cause significant brain damage and has been linked with an increased risk of d

ABSTRACT Chlorpyrifos (CPF) is a widely used organophosphate insecticide. In addition to its known properties of choline

BACKGROUND AND PURPOSE: Nebivolol, a selective beta(1)-adrenoceptor antagonist mediating rapid vasodilating effect

Although oxidative stress is fundamental to the etiopathology of Parkinson disease, the signaling molecules involved in tr

Reactive oxygen species (ROS) actively participate in microglia-mediated pathogenesis as pro-inflammatory molecules. H

When neural cells are exposed to paraquat, nitric oxide generation increases primarily due to an increase in the expressi

Paraquat (PQ) is a well-known herbicide that exerts its effects by elevating intracellular levels of superoxide. It has been j

The effects of cannabinoids in mitochondria after acute oxidative stress insult are not fully established. We investigated t

Mitochondrial reactive oxygen species (ROS) play an important role in both physiological cell signaling processes and nun

This study aims to elucidate the processes underlying neuroprotection of kaempferol in models of rotenone-induced acu

Rasagiline is a propargylamine and irreversible monoamine oxidase (MAO) B inhibitor used for the treatment of Parkinso

Talipexole is a non-ergot dopamine (DA) agonist that has been used in the treatment of Parkinson's disease. In the prese

Triple A syndrome is named after the main symptoms of alacrima, achalasia, and adrenal insufficiency but also presents v

Paraquat produces dopaminergic pathologies of Parkinson's disease, in which cyclooxygenase-2 (COX-2) is implicated. Hc

BACKGROUND: Mutations in Parkin are the most common cause of autosomal recessive Parkinson disease (PD). The mitc

Apoptosis signal-regulating kinase 1 (ASK1) is activated by various types of stress, including, endoplasmic reticulum (ER) s

Mitochondrial dysfunction has been implicated in the pathogenesis of a number of neurodegenerative disorders includin

trans-Resveratrol (RES) is one of a number of dietary polyphenols that have been reported to beneficially affect human p

There are two causes of Parkinson's disease (PD): environmental insults and genetic mutations of PD-associated genes. E

Exposure to paraquat (PQ; N,N'-dimethyl-4,4'-bipyridium), a potent herbicide, can lead to neuronal cell death and increa

Oxidative stress plays pivotal roles in aging, neurodegenerative disease, and pathological conditions such as ischemia. W

Epidemiological and in vivo studies have demonstrated that exposure to the pesticides paraquat (PQ) and maneb (MB) in

Mitochondrial oxidative stress is a contributing factor in the etiology of numerous neuronal disorders. However, the prec

OBJECTIVE: To investigate the effect of paraquat on induction of cell damage and miR-133b expression in PC12 cells. MET

Previously, we have reported that a 10-amino acid peptide (MIIYRDLISH) derived from the NH(2)-terminus of the human

BACKGROUND: Neuronal iron accumulation is thought to be relevant to the pathogenesis of Parkinson's disease (PD), alt

Environmental neurotoxic exposure to agrochemicals has been implicated in the etiopathogenesis of Parkinson's disease

Paraquat is a cationic herbicide that causes acute cell injury by undergoing redox cycling. Oxidative stress is thought to be

BACKGROUND: TDP-43 proteinopathies are characterized by loss of nuclear TDP-43 expression and formation of C-termir

OBJECTIVE: To investigate the protective effects of the tert-butylhydroquinone (tBHQ) pretreatment on neurotoxicity and

Parkinson's disease (PD) is one of the most common age-related neurodegenerative diseases and affects millions of peop

Paraquat is a widely used herbicide that is structurally similar to the known dopaminergic neurotoxicant 1-methyl-4-pher

AIMS: Delta(9)-tetrahydrocannabinol (Delta(9)-THC) is neuroprotective in models of Parkinson's disease (PD). Although C

Paraquat (PQ) was demonstrated to induce dopaminergic neuron death and is used as a Parkinson's disease (PD) mimeti

P-ATPases are membrane transporters energized by ATP. The subfamily of P(5)-ATPases is the least studied P-ATPases an

BACKGROUND AND PURPOSE The mechanisms of paraquat (PQ)-induced toxicity are poorly understood and PQ poisonin

TDP-43 proteinopathies are characterized by loss of nuclear TDP-43 and accumulation of the protein in the cytosol as ubi

In this study, we have compared several features of cell death triggered by classical inducers of apoptotic pathways (etop

In vitro and in vivo models of Parkinson's disease (PD) suggest that increased oxidant production leads to mitochondrial c

AIMS: Chronic exposure to environmental toxicants, such as paraquat, has been suggested as a risk factor for Parkinson's

Amyotrophic lateral sclerosis (ALS) is a progressive, fatal, motor neuron disease with no effective long-term treatment of

Mitochondria are considered major generators of cellular reactive oxygen species (ROS) which are implicated in the path

Three types of resveratrol analogues were designed, synthesized and evaluated in vitro against paraquat-induced apopto

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Reactive oxygen species (ROS) function as modulators of pro-inflammatory processes in microglia-associated neurodegeneration. The loss of dopaminergic neurons induced by the parkinsonian toxins paraquat, rotenone, and 1-methyl-4-phenylpyridinium (MPP+) is a major cause of Parkinson's disease (PD). Paraquat (PQ) is a neurotoxic herbicide that induces superoxide formation. Although it is known that its toxic properties are mediated by ROS, the exact mechanism of action remains unclear. Paraquat (PQ), a cationic nonselective bipyridyl herbicide, has been used as neurotoxicant to modulate Parkinson's disease in animal models. Neural progenitor cells (NPCs) derived from human embryonic stem cells (hESCs) have great potential in cell therapy, drug discovery, and disease modeling. Our previous works have shown that the (NADPH) oxidase (Nox) enzyme, in particular Nox1, plays an important role in oxidative stress and neurodegeneration. Controversial reports on the role of autophagy as a survival or cell death mechanism in dopaminergic cell death induced by MPP+ and PQ. OBJECTIVE: To investigate effects of paraquat on the mRNA expression of key elements of Notch signaling (Notch1, Jagged1, and Numb). Paraquat (PQ) is one of the most widely used herbicides in the world. Although available evidence indicates that people exposed to PQ are at a higher risk of developing Parkinson's disease, the exact mechanism of action remains unclear. Paraquat (PQ) is a widely used, highly toxic and non-selective contact herbicide, which has been associated with central nervous system (CNS) damage. The neurotoxin paraquat (PQ) causes apoptosis of dopaminergic neurons in mammalian cell culture and animal models, and is used as a Parkinson's disease (PD) mimetic. OBJECTIVE: To investigate the effects of paraquat on microRNA expressions in PC12 cells, and to explore the regulatory mechanisms of microRNAs in PD. Recent studies suggest that traumatic brain injury (TBI) and pesticide exposure increase the risk of Parkinson's disease (PD). Mitochondrial reactive oxygen species are implicated in the etiology of multiple neurodegenerative diseases, including PD. The developmental and stress-regulated alternative TrkAIII splice variant of the NGF receptor TrkA is expressed by advanced stages of PD. Parkinson's disease (PD) is a multifactorial disorder with a complex etiology including genetic risk factors, environmental factors, and aging. Aberration during the development of the central nervous system (CNS) due to environmental factors underlies a variety of neurodegenerative diseases. It has been suggested that glial cells in the central nervous system might function as a buffer and protect neurons and synapses. Omega-3 polyunsaturated fatty acid levels are reduced in the substantia nigra area in Parkinson's disease patients and are associated with disease severity. The olfactory bulb is one of the most vulnerable brain regions in age-related proteinopathies. Proteinopathic stress is mitigated by the olfactory bulb. Paraquat (PQ) as a Parkinsonian mimetic has been demonstrated to impair dopaminergic (DAergic) neurons and is highly neurotoxic. Lithium is a mood stabilizing agent commonly used for the treatment of bipolar disorder. Here, we investigated the potential of lithium to protect against PQ-induced neurotoxicity. Paraquat (PQ) was demonstrated to induce dopaminergic neuron death and is used as a Parkinson's disease (PD) mimetic in animal models. Intracytoplasmic inclusions of protein aggregates in dopaminergic cells (Lewy bodies) are the pathological hallmark of Parkinson's disease. Oxidative stress or reduced expression of naturally occurring antioxidants during aging has been identified as a major cause of neurodegeneration. The mechanism of intracellular metabolism of methylmercury (MeHg) is not fully known. It has been shown that superoxide anion and hydrogen peroxide are involved in the metabolism of MeHg. Oxidative stress and the ubiquitin-proteasome system play a key role in the pathogenesis of Parkinson disease. Although the exact mechanism of action remains unclear, compelling evidences have shown that diverse environmental insults arising during early life can either directly lead to a neurodegenerative disorder or increase the risk of developing Parkinson's disease. Carnosic acid (CA) is a phenolic diterpene isolated from *Rosmarinus officinalis* and exerts anti-inflammatory, antioxidant, and neuroprotective effects. Herbicides containing paraquat may contribute to the pathogenesis of neurodegenerative disorders such as Parkinson's disease. Paraquat (PQ) exposure influences central nervous system and results in serious neurotoxicity in vitro and in vivo. However, the exact mechanism of action remains unclear. OBJECTIVE: To investigate the role of Wnt signaling pathway on paraquat (PQ)-induced PC12 cells damage. METHODS: Using Western blotting and RT-PCR. Parkinson's disease (PD) is a neurodegenerative disorder characterised by the loss of dopaminergic neurons in the substantia nigra. OBJECTIVE: To explore the effect of paraquat (PQ) on autophagy in human embryonic neural progenitor cells. METHODS: Using Western blotting and RT-PCR. While environmental exposures are not the single cause of Parkinson's disease (PD), their interaction with genetic alterations may increase the risk of developing PD. Tanshinone I (T-I; 1,6-Dimethylnaphtho[1,2-g][1]benzofuran-10,11-dione; C₁₈H₁₂O₃), which may be found in *Salvia miltiorrhiza*, has been shown to protect against PD. *Journal of Neurochemistry*. 2007. 102:55-56

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The protective effect of tyrosol on apoptosis in PC12 cell induced by paraquat (PQ) was studied. The PC12 cell was cultured in the presence of tyrosol. A combination of the herbicide paraquat (PQ) and fungicide maneb (MB) has been linked to Parkinson's disease. Previous studies have shown that both MPTP and MPP+ inhibited the NADPH-dependent microsomal LPO in mouse brain and lung. On the other hand, PQ and MB have been shown to induce LPO in mouse brain and lung. The effect of 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP), 1-methyl-4-phenylpyridinium (MPP+) and 1,1-dimethyl-4-phenylpyridinium (DMPP+) on LPO in mouse brain and lung was also studied. We had previously reported that 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP), which produces Parkinson's disease in mice, is a potent inducer of LPO in mouse brain and lung. The effects of 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP), its metabolite 1-methyl-4-phenyl pyridinium ion (MPP+), and 1,1-dimethyl-4-phenylpyridinium (DMPP+) on LPO in mouse brain and lung were also studied. Glucocorticoids (GCs) disrupt the energy metabolism of neurons of the hippocampus, and thus leave them more vulnerable to oxidative stress. This study reports the effects of Ca²⁺ channel blockers (Ca antagonists) on intraneuronal Ca²⁺ ([Ca²⁺]_i) movements and the effects of 1-Methyl-4-phenylpyridinium (MPP+), the active metabolite of 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP) on intraneuronal Ca²⁺ ([Ca²⁺]_i) movements.

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Organotypic cultures of the brain provide a unique opportunity to directly examine the regional vulnerability of specific brain regions. Paraquat has been implicated as an environmental toxin which may induce the syndrome of Parkinson's disease after exposure. Exposure to pesticides may be a risk factor for Parkinson's disease based on epidemiologic data in humans, animal models, and cell cultures. Paraquat (1,1'-dimethyl-4,4'-bipyridinium, PQ) is a herbicide that is thought to possibly induce Parkinson's disease (PD), since a strong correlation has been found between paraquat exposure and the development of PD. Glutamate-mediated excitotoxicity might contribute to the pathogenesis of Huntington's disease and other polyglutaminopathies. Environmental exposure to the oxidant-producing herbicide paraquat has been implicated as a risk factor in Parkinson's disease. Mutations in Cu/Zn-superoxide dismutase 1 (SOD1) are responsible for a familial form of amyotrophic lateral sclerosis (FALS). A loss of nigrostriatal dopaminergic neurons is the primary neurodegenerative feature of Parkinson's disease. Paraquat, a potent herbicide, has been implicated as a potential risk factor for the development of Parkinson's disease. In experimental animals, exposure of mice to the herbicide paraquat has been demonstrated to result in the selective loss of dopaminergic neurons. Paraquat, MPTP, and rotenone reproduce features of Parkinson's disease (PD) in experimental animals. The exact mechanism of action of these agents is not clear. Nonsteroidal anti-inflammatory drugs (NSAIDs) have been shown to amplify the heat shock response in cell lines by increasing the expression of heat shock proteins. In recent years, several lines of evidence have shown an increase in Parkinson's disease (PD) prevalence in rural environments. Paraquat (PQ) is a cationic nonselective bipyridyl herbicide widely used to control weeds and grasses in agriculture. Epidemiological studies have shown a strong correlation between paraquat exposure and the development of Parkinson's disease (PD). Parkinson's disease (PD) is a common neurodegenerative disorder and is characterized by the progressive loss of dopaminergic neurons in the substantia nigra. Parkinson disease (PD) is a common neurodegenerative disorder characterized by the progressive loss of dopaminergic neurons in the substantia nigra. The orphan nuclear receptor Nurr1 is required for the development of the ventral mesencephalic dopaminergic neurons. Paraquat, N-methyl-4-phenyl-1,2,3,6 tetrahydropyridine, and rotenone have been shown to reproduce several features characteristic of Parkinson's disease. Toxic concentrations of paraquat (0.2mM, 24h) caused death of both mature and immature cerebellar granule neurons (CGNs). Paraquat (PQ) is suspected to be an environmental risk factor for Parkinson's disease (PD). A strong correlation between paraquat exposure and the development of Parkinson's disease (PD) has been reported. Paraquat (PQ) causes selective degeneration of dopaminergic neurons in the substantia nigra pars compacta, reproducing the pattern of neuronal loss seen in Parkinson's disease. An important feature of Parkinson's disease is the degeneration of dopaminergic neurons in the Substantia Nigra pars compacta. Parkinson's disease (PD) is a neurodegenerative disease that mainly affects dopaminergic (DA-ergic) neurons in the substantia nigra. Inhibition of mitochondrial complex I is one of the leading hypotheses for dopaminergic neuron death associated with Parkinson's disease. BACKGROUND: Oxidative stress (OS) is an important factor in brain aging and neurodegenerative diseases. Certain neurodegenerative diseases are characterized by increased oxidative stress. Oxidative stress is the common downstream effect of a variety of environmental neurotoxins that are strongly implicated in the pathogenesis of neurodegenerative diseases. Both epidemiological and pathological data suggest an inflammatory response including microglia activation and neuroinflammation in Parkinson's disease. Axonal degeneration is a common pathologic feature in peripheral neuropathy, neurodegenerative disease, and normal aging. Mechanistic studies underlying dopaminergic neuron death may identify new drug targets for the treatment of Parkinson's disease. The effects of the 1-methyl-4-phenylpyridinium ion (MPP(+)) and some structurally related compounds on mitochondrial function and cell death have been studied. Parkinson's disease (PD) has been linked to exposure to a variety of chemical (e.g., pesticides) and inflammatory agents, and to genetic factors. The study of glial derived factors induced by injury and degeneration is important to understand the nervous system response to neurodegeneration. We have previously demonstrated that alpha-synuclein overexpression increases the membrane conductance of dopaminergic neurons. Investigation of mechanisms responsible for dopaminergic neuron death is critical for understanding the pathogenesis of Parkinson's disease. The herbicide paraquat (PQ) has increasingly been reported in epidemiological studies to enhance the risk of developing Parkinson's disease. Primary cultures of fetal rat cortical neurons and astrocytes were used to test the hypothesis that astrocyte-mediated oxidative stress contributes to the neurotoxicity of paraquat. This study determined how preconditioned neurons responded to oxygen-glucose deprivation (OGD) to result in neuronal death. The present study was aimed at determining the role of paraquat (PQ) in the activation of the NF-E2-related factor 2 (Nrf2) pathway. The neurotoxins paraquat (PQ) and dopamine (DA or 6-OHDA) cause apoptosis of dopaminergic neurons in the substantia nigra. Accumulation of misfolded alpha-synuclein is the pathological hallmark of Parkinson's disease (PD). Nevertheless, little is known about the mechanisms underlying the pathogenesis of PD. Recent findings implicate the calcium-permeable transient receptor potential (TRP) melastatin subtype 2 (TRPM2) and calcium in the pathogenesis of PD. The two hit hypothesis of neurodegeneration states that cells that have been severely stressed once are more vulnerable to subsequent challenges. Xenobiotic exposure is a risk factor in the etiology of neurodegenerative disease. It was recently hypothesized that restriction of mitochondrial function by paraquat is a key event in the pathogenesis of Parkinson's disease. The herbicide paraquat is an environmental factor that may be involved in the etiology of Parkinson's disease (PD). Systemic exposure to paraquat in experimental animals increases the risk of developing Parkinson's disease. Epidemiological and animal studies suggest that environmental toxins including paraquat (PQ) increase the risk of developing Parkinson's disease. The dual-hit hypothesis of neurodegeneration states that severe stress sensitizes vulnerable cells to subsequent challenges. Paraquat-stimulated NADPH-dependent lipid peroxidation in mouse brain and pulmonary microsomes was inhibited by superoxide dismutase and catalase.

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Paraquat stimulates NADPH-Fe(2+)-dependent microsomal lipid peroxidation in mouse brain and strongly inhibits it in th

The cytotoxicity of reactive oxygen species and related agents toward cultured rat adrenal medullary phenochromocytom

We have investigated the response to oxidative stress in a model system obtained by stable transfection of the human ne

In the central nervous system oxidative stress has been implicated in the pathology of several neurological disorders. The

The brain is particularly vulnerable to oxygen free radicals, which have been implicated in the pathology of several neuro

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Nitric oxide synthase (NOS) oxidizes L-arginine to NO(&z.ccirf;) and L-citrulline. Recent studies have shown that this enzy

Paraquat stimulated NADPH-dependent lipid peroxidation in mouse brain microsomes, while it suppressed lipid peroxide

BACKGROUND: An increase in reactive oxygen species (ROS) burden and subsequent oxidative damage to nucleic acids, p

Carnosic acid (CA; C₂₀H₂₈O₄), which is also called salvini, is a major phenolic diterpene found in *Rosmarinus officinalis* L.

Pinocembrin (PB; 5,7-dihydroxyflavanone; C₁₅H₁₂O₄) is a flavonoid found in propolis and exerts antioxidant, anti-inflam

ETHNOPHARMACOLOGICAL RELEVANCE: Parkinson's disease (PD) is a multifactorial neurodegenerative disorder affecting

Paraquat is a neurotoxic agent, and oxidative stress plays an important role in neuronal cell death after paraquat exposur

Rat cultured cerebellar granule neurons (CGNs) were not sensitive to CuCl₂ (1-10 microM, 24 h), whereas paraquat (150 i

Mitochondrial dynamics and quality control plays a critical role in the maintenance of mitochondrial homeostasis and fur

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Mitochondrial oxidative stress is a contributing factor in the etiology of numerous neuronal disorders. However, the prec

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Cells from the midbrain micromass cell culture system from rat day 13 embryos were used to investigate the developme

Objective To evaluate the protective effect of peanut sprout extract (PSE) against paraquat (PQ) induced SK-N-SH cells. M

Purpose: To investigate whether two synthetic prenylated xanthone analogues -1,3,6,8-tetrahydroxy-9H-xanthen-9-one (

Oxidative stress caused by an increase in free radicals plays an important role in neuronal death. We investigated the eff

Oxidative stress and apoptosis play pivotal roles in the pathogenesis of neurodegenerative diseases. We investigated the

Background and purpose: Resveratrol (RSV) is a naturally existing polyphenolic compound abundantly found in grapes an

Paraquat (PQ) through electron transfer reactions with NADH-dependent oxidoreductase of mitochondria and NADPH-de

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1. Objectives Mutations in the PINK1 gene are responsible for autosomal recessive Parkinson's disease (PD). The project i

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